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Genetic Mapping of Sheath Blight Resistance QTLs within Tropical *Japonica* Rice Cultivars

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ABSTRACT

Most commercial cultivars of rice (*Oryza sativa* L.) are susceptible to sheath blight (SB), a devastating fungal disease causing significant losses in grain yield and quality. There are limited sources of genetic resistance adapted to U.S. growing conditions, and no commercial long grain cultivar of rice is currently available in the United States with a high level of SB resistance. Sheath blight resistance has been reported to be horizontal and quantitative. A population of 279 $F_{2:3}$ progeny rows derived from a cross between two tropical *japonica* U.S. rice cultivars, Rosemont (semi-dwarf, SB susceptible) and Pecos (tall, SB resistant), was used to map SB resistance. Progeny families were evaluated for disease reactions, plant height (PH), and heading date (HD) in replicated field trials for 2 yr and genotyped with 149 simple sequence repeat markers. Correlation analysis between SB ratings with PH and HD showed that both agronomic traits were significantly correlated with SB resistance. Four significant (logarithm of odds ratio ≥ 3.6) quantitative trait loci (QTLs) were identified for SB resistance, with individual effects explaining 5.6 to 33.4% of the total phenotypic variation. Plant height appears to have a direct influence on SB resistance, with QTLs for these traits colocated on chromosome 1. Consistent results across years indicate the stability of the identified QTLs and their potential for improving rice SB resistance using marker-assisted selection.

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Abbreviations: CIM, composite interval mapping; HD, heading date; IM, interval mapping; LOD, logarithm of odds (LOD); MIM, multiple interval mapping; MQM, multiple QTL model; PH, plant height; PVE, phenotypic variation explained; QTL, quantitative trait locus; SB, sheath blight; SSR, simple sequence repeat.

Sheath blight, caused by the fungus *Rhizoctonia solani* Kühn, is one of the most important diseases of rice (*Oryza sativa* L.), causing severe loss in grain yield and quality worldwide (Lee and Rush, 1983; Rush and Lindberg, 1996). *Rhizoctonia solani* is a semi-saprophytic fungus with a broad host range, affecting many crops including rice, maize (*Zea mays* L.), wheat (*Triticum aestivum* L.), sorghum [*Sorghum bicolor* (L.) Moench], common bean (*Phaseolus* spp.), and soybean [*Glycine max* (L.) Merr.] (Zhao et al., 2006). No commercial long-grain cultivar of rice is currently available in the United States having a high level of SB resistance. However, rice cultivars with significantly different levels of SB resistance have been observed in international germplasm (Khush, 1977; Guo et al., 1985; Groth and Novick, 1992). Rice SB resistance is generally believed to be a typical quantitative trait controlled by several genes (Sha and Zhu, 1989; Li et al., 1995b). On the other hand, a few studies (Xie et al., 1992; Pan et al., 1999a,b) proposed that SB resistance in some rice varieties is controlled by a few major genes. The identification of genes that affect complexly inherited traits is often difficult and is best approached through developing

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a genetic linkage map to identify quantitative trait loci (QTLs) (Tanksley and McCouch, 1997).

Several studies (Groth and Novick, 1992; Hashiba et al., 1981; Li et al., 1995a) have reported associations between SB resistance and agronomic traits, such as plant height (PH) and heading date (HD). Such confounding factors, together with the complex genetic nature of resistance, have contributed to a limited success in breeding for SB resistance using conventional methods. Weather conditions, cultural management practices, and planting of susceptible varieties can result in widespread losses due to this disease. At present, application of preventive fungicides is the most common control measure for SB disease, which increases production costs and presents an environmental risk. Thus, rice breeders are seeking SB-resistant, semidwarf cultivars with early maturity and high yield potential. New strategies are needed for the identification, validation, and effective transfer of genes for SB resistance into rice cultivars.

Sheath blight resistance QTLs have been previously reported in rice (Li et al., 1995b; Zou et al., 2000; Pinson et al., 2005). Each of these previous studies identified SB resistance in crosses between *O. sativa* ssp. *indica* and ssp. *japonica*, primarily focusing on genes associated with the higher degree of SB resistance more frequently found in *indica* rice germplasm. However, most *indica* rice is poorly adapted for production in the United States, and limited gains have been made on introgressing *indica* germplasm into the *japonica* rice primarily grown in the United States. (Mackill and McKenzie, 2003; Lu et al., 2005; Eizenga et al., 2006). Because U.S. rice breeders can be reluctant to use *indica* germplasm in their breeding programs, we wished to identify SB resistance genes present in *japonica* germplasm that is already adapted for U.S. rice production.

MATERIALS AND METHODS

Plant Materials and Mapping Population

The mapping population analyzed consisted of 279 $F_{2:3}$ progeny lines derived from a cross between two U.S. tropical *japonica* rice cultivars, Rosemont and Pecos. Rosemont (PI 546365; pedigree of CIor9881/PI331581//L-201) is a very early-maturing, long-grain, semidwarf cultivar that is highly susceptible to *R. solani* (Bollich et al., 1993). Pecos (PI 476818; pedigree of CIor9545//'Gulfrose'/'Tainan Iku 487') is an early-maturing, medium-grain, tall cultivar with notable resistance to SB (Bollich et al., 1985; summarized 1982 to 1989 SB disease readings from U.S. Uniform Regional Rice Nursery data). The $F_{2:3}$ seed samples were divided into subsamples to support replicated field evaluation of SB response, PH, and HD over 2 yr.

Field Evaluation of Sheath Blight and Agronomic Traits

The 279 $F_{2:3}$ families were evaluated in field trials conducted in 2002 and 2003 at the Texas A&M University Agricultural Research and Extension Center in Beaumont, TX. Each year, the study was conducted using randomized complete block design having two replications. The $F_{2:3}$ families were drill-seeded in two-row plots 2.4-m long with 18 cm between rows. To standardize border effects, each test plot was separated by a single row of Rosemont (susceptible parent) planted at the same row spacing. Field plots were inoculated approximately 60 d after planting with approximately 100 mL of inoculum per plot of a 2:1 (v:v) mixture of rice hulls and unhulled grains infested with the pathogen (Marchetti and Bollich, 1991). Overhead sprinklers simulating morning dew and afternoon rain showers were used to promote ideal conditions for pathogen growth and disease development.

Disease scores were determined approximately 30 d after heading for each plot (Marchetti and Bollich, 1991). The maximum disease score observed among the two replications was considered the final disease rating for each year to avoid data bias due to random susceptible plots escaping disease, which is a common problem in disease evaluations. The SB response rating system used was a 0 to 9 scale, with 0 indicating no evidence of infection and 9 indicating plants completely killed due to disease (Marchetti and Bollich, 1991). Each unit of the scale approximated the above water proportion of the plants, in tenths, that exhibit necrotic disease lesions (e.g., a rating of 5 indicates that approximately 50 to 59% of the plant area above the water line exhibited necrotic lesions).

The agronomic traits PH and HD were measured to evaluate their relationship with SB resistance. Plant height (in centimeters), measured from the soil surface to the tip of the tallest panicle at maturity, was recorded for each plot. Heading date (in days) was recorded as the number of days between sowing and the point at which 50% of plants within the plot had at least one panicle flowering.

Molecular-Marker Assays

Flag leaf tissue was collected for DNA isolation from each of the original F_2 plants from which the F_3 progeny row seed was derived, along with both parental lines. Total genomic DNA was isolated following the method of Fjellstrom et al. (2004). Parental marker polymorphisms were tested using 808 simple sequence repeat (SSR) markers chosen based on their containing 15 or more microsatellite repeats (McCouch et al., 2002). To provide uniform genome coverage along the 12 rice chromosomes and to minimize marker location redundancy, 149 polymorphic SSR markers were selected on the basis of their previously mapped locations using the Gramene website database (www.gramene.org). Simple sequence repeat markers were amplified following the techniques of Fjellstrom et al. (2004). Simple sequence repeat primer sequences were obtained from the Gramene website, except for Con673 (forward primer sequence = CGT ACT TGC CAC CGT AAG; reverse = TTG ATA GGC AAT GTT TCT CC). The fluorescently labeled amplification products of the mapping population were analyzed after separation on a 6% PAGE sequencing gel using a Li-Cor 4200 sequencing system (LI-COR Inc., Lincoln, NE) or an ABI 3100 Genetic Analyzer capillary gel electrophoresis system (Applied Biosystems, Foster City, CA). Both systems allowed multiplexing of differently labeled primer pairs, facilitating high-throughput genotypic data generation.

Mapping and Statistical Analyses

A genetic map was constructed for the genotyped F_2 population using JoinMap 3.0 (Van Ooijen and Voorrips, 2001). Genetic

distance was expressed in centimorgans using the Kosambi mapping function (Kosambi, 1944). StatView 5.0.1 (SAS Institute, Cary, NC) software was used for correlation and regression analyses. The distributions of SB scores for the progeny lines in 2002 and 2003 were plotted and presented separately, while the PH and HD distributions were plotted and presented as the mean of the 2-yr data. Simple sequence repeat allele data of Garris et al. (2005) and Lu et al. (2005) used in marker diversity analyses were obtained from the Gramene website genetic diversity database (http://www.gramene.org/db/diversity/diversity_view).

Quantitative trait loci were identified and validated using two analytical approaches and software programs: interval mapping (IM) using MapQTL 5.0 (Van Ooijen, 2004) and composite interval mapping (CIM) using Windows QTL Cartographer v. 2.5 (Wang et al., 2005; http://statgen.ncsu.edu/qtlcart/WQTLCart.htm). Both programs were used to run 1000 permutations to calculate appropriate threshold logarithm of odds (LOD) ratio scores (Lander and Botstein, 1989) for declaring a QTL at a significance level $P < 0.05$ for each trait (Churchill and Doerge, 1994). All LOD threshold values were found to be quite similar, ranging only from 3.4 to 3.7. We elected to use a standard LOD threshold of 3.6 to provide a better comparison between the analytical methods and traits. Using IM, when the LOD score exceeded 3.6, the position with the highest LOD score on each chromosome was estimated as the most likely position of a QTL. The MapQTL program was also used to obtain estimates of the percentage of the total phenotypic variation explained (PVE) and the phenotypic effects by each QTL.

The second approach used for QTL identification was CIM analysis (Zeng, 1994), which employed model 6 of Windows QTL Cartographer v. 2.5 (Wang et al., 2005). Automatic cofactor selection using a forward–backward regression was performed using five control markers, a walking speed of 2 cM, and an LOD threshold of 3.6. Quantitative trait loci identified from CIM were further tested and newly significant QTLs identified using multiple interval mapping (MIM; Kao et al., 1999) as conducted within QTL Cartographer v. 2.5, which evaluates the existence of additional QTL with the effect of the CIM-identified QTLs set into the model. The significance criteria for naming additional QTLs in MIM were of the Bayesian Information Criteria Model 0 (BIC-M0). Multiple interval mapping was used to further evaluate the additive (main) genetic effects of the QTLs and any QTL × QTL epistatic effects. The R^2 values from the resulting MIM genetic models for each phenotypic trait were accepted as the percentage of PVE by the identified QTLs. Additional QTLs were also sought using the multiple QTL model (MQM) of MapQTL. Similar to the CIM analysis shown above, MQM identifies multiple QTLs by regression analysis, with removing the effect of major individual QTLs revealed during IM by manually selecting them as cofactors. The MQM results are not shown, but were used to validate the reported CIM results.

Table 1. Means and ranges in $F_{2:3}$ progeny rows and parental means for sheath blight (SB) resistance, plant height and heading date.

Trait	Progeny			Parent mean	
	Mean	Range	CV	'Rosemont'	'Pecos'
SB resistance, 0–9†	6.2	2.0–9.0	0.23	8.1	2.1
Plant height, cm	107.7	81.3–133.5	0.10	94.0	115.0
Heading date, d	81.8	73.8–90.3	0.04	78.5	85.1

†Sheath blight disease score; 0 = no disease symptoms, 9 = plants killed because of disease (Marchetti and Bollich, 1991).

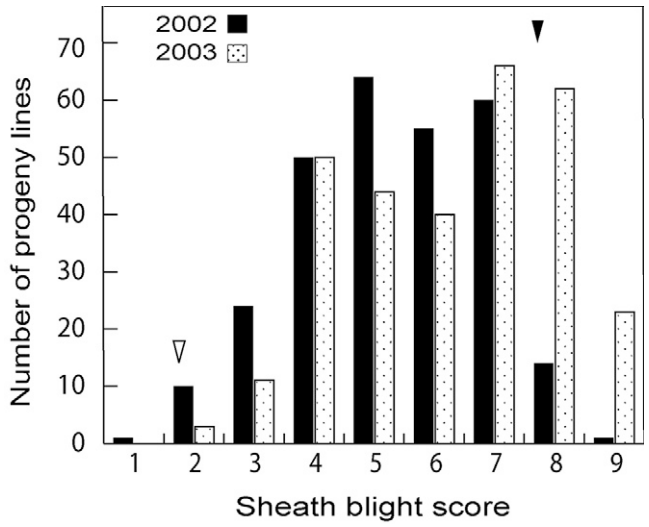

Figure 1. Distribution of 279 $F_{2:3}$ progeny lines for sheath blight disease scores, based on a 0 to 9 rating scale, shown separately for 2002 and 2003. The mean trait values for the parents of the progeny lines, 'Pecos' and 'Rosemont', are indicated as hollow and filled arrows, respectively.

RESULTS AND DISCUSSION

Phenotype Distribution Analysis

The $F_{2:3}$ progeny population exhibited significant phenotypic variance for all traits to support QTL mapping (Table 1). The progeny SB disease scores were continuously distributed, as expected for a quantitative trait. However, disease severity was greater in 2003 than 2002, as demonstrated by the skewed distribution of SB scores in the second year of testing (Fig. 1). The distribution of family PH and HD (Fig. 2) are presented as means because their measurements were consistent across replications and years. The distribution for PH was bimodal (Fig. 2A), having distribution peaks at 100 cm and 115 cm in height, suggesting the involvement of a single gene with large effects controlling this agronomic trait. In contrast, the normal distribution of HD indicated polygenic segregation of this trait (Fig. 2B).

Segregation of Markers in the $F_{2:3}$ Population

Of the 808 SSR markers screened for parental polymorphism, 638 were successfully amplified for both parents and 348 (54.5%) were polymorphic in the population. Segregation of the 149 mapped SSR marker loci in the 279 $F_{2:3}$ progeny were tested for fit against a Mendelian ratio of 1 homozygous Rosemont:2 heterozygous:1 homozygous Pecos, as expected in an F_2 population. The $F_{2:3}$ population was homozygous Rosemont for 25.5%, homozygous Pecos for 23%, and heterozygous for 47.4% of the markers. The chi-square tests of the frequencies of individual parental alleles in the $F_{2:3}$ population indicated that 29 of 149 mapped

marker loci (20%) displayed significant deviation from the expected 1:2:1 ratio, with a maximum of 121 homozygous Rosemont alleles (43% of progeny) seen for marker RM566 on chromosome 9 (data not shown). The moderate amount of segregation distortion found in this mapping population should not greatly affect QTL analyses, and no additional steps were made to take this distortion into account. In most cases, the allelic frequency distortions favored alleles from the Rosemont parent, suggesting that increased Rosemont allele frequencies were associated with increased fitness. For example, Rosemont alleles of three linked loci on chromosome 1 and seven linked loci on chromosome 9 were more prevalent (data not shown) and associated with two QTLs affecting PH (see following sections). Segregation distortion of alleles at marker loci has been reported in previous studies in rice (Wang et al., 1993; Li et al., 1995a).

Linkage Map Construction

A genetic linkage map was constructed based on the 149 SSR markers in the population of 279 F_2 progeny lines. This map spanned approximately 1465 cM of the 12 rice chromosomes, with an average marker distance of 9.8 cM between markers (Fig. 3). The chromosome placement and order for 147 of the SSRs was in good agreement with the IRMI-2003 map (available at http://www.gramene.org/). However, markers RM14 and RM6732 were placed on chromosomes 1 and 11, respectively, while they were mapped to chromosomes 8 and 12 on the IRMI-03 map, respectively, as well as in BLAST sequence searches. It is not clear why these markers map to different chromosomes in our study, although RM6732 also appears to map to chromosome 11 in another genetic cross we are presently studying. The number of markers per chromosome ranged from 9 (chromosome 7, 10) to 22 (chromosome 3), with an average of 12 markers per chromosome. Chromosomes 1 and 3 were the longest linkage groups, whereas chromosomes 10 and 12 were among the shortest, consistent with other previously developed linkage maps of rice (http://www.gramene.org/). Only a few chromosomes contained intervals 20 cM or larger (Fig. 3). Two separate sections of chromosomes 1, 5, and 7 could not be joined due to insufficient linkages. This could be due to lack of polymorphism spanning those regions in this *japonica/japonica* population.

QTLs for Sheath Blight Resistance

Four significant (LOD ≥ 3.6) SB resistance QTLs were identified on chromosomes 1, 2, 3, and 9 and individually explained 5.8 to 36.4% of the phenotypic variation (Table 2, Fig. 3). Except for the QTL on chromosome 2, the resistant parent, Pecos, contributed the resistance QTLs. All four resistance QTLs were identified in both years (2002, 2003) with IM as well as CIM approaches. The resistance QTL on the long arm of chromosome 1 (lower end of chromosome in Fig. 3) had the largest effect, explaining 36.4% of the phenotypic variation (LOD = 25.2), with a QTL peak at marker RM1339. A fifth subthreshold SB resistance QTL (LOD = 3.1) on chromosome 7, peaking at RM1362, was identified with CIM only in 2003, whereas the same QTL was not detected with IM. Detection of this QTL in 2003 is likely due to higher disease development in 2003. Sheath blight resistance QTLs have been reported at similar genomic locations in previous studies (Li et al., 1995b; Zou et al., 2000; Pinson et al., 2005), except for the QTL on chromosome 1, which, to our knowledge, has not been reported before. Recognizing that the SB resistance alleles at the QTL regions on chromosomes 2, 3, and 9 were previously identified as originating in *indica* germplasm, it is important to note that the resistance alleles identified at the same QTL regions in this study appear to provide sources of SB resistance from *japonica* germplasm as well. Independent genetic distance analyses of SSR loci (Garris et al., 2005; Lu et al., 2005) indicate that Pecos and its progenitors (Gulfrose, 'Rexark Rogue', and Tainan Iku 487; Bollich et

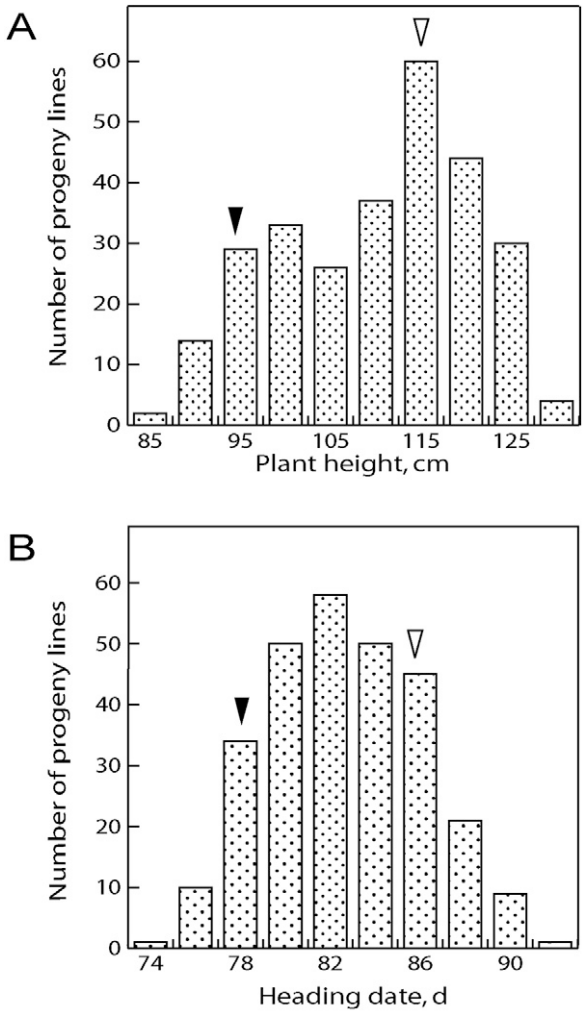

Figure 2. Distributions of 279 $F_{2:3}$ progeny lines for combined two-year averages of (A) plant height and (B) heading date. The mean trait values for the parents of the progeny lines, 'Pecos' and 'Rosemont', are indicated as hollow and filled arrows, respectively.

al., 1985) are grouped within *japonica* germplasm, thus confirming that these are unique *japonica* SB resistance alleles not previously reported.

QTLs for Plant Height and Heading Date

Two separate QTLs for PH were mapped on chromosomes 1 and 9 (Table 2, Fig. 3). The PH QTL with largest effect was on chromosome 1. This QTL individually accounted for 77.0% of the variation in PH, with an additive effect of approximately 13.8 cm for increased height. The identification of major QTLs for PH is not surprising, as the difference in PH between the parents was 21 cm, with a range of 81.3 to 133.5 cm between lines in the progeny population (Table 1). Furthermore, the semidwarf gene *sd-1* is known to be located near RM1339 on chromosome 1, where the largest QTL peak for PH was found. A similar QTL for PH has been reported by Yu et al. (2002). The other QTL for PH, on chromosome 9, was detected only in CIM. Both QTLs for increased height were contributed by the tall parent, Pecos.

Two QTLs for HD were identified on separate regions of chromosome 3. The HD QTL on top of chromosome 3 was a major QTL, explaining 29.7% of variation in HD, with the highest LOD at marker RM3894 (Table 2, Fig. 3). Previous studies (Yu et al., 2002; Pinson et al., 2005) also reported QTLs for HD in the similar location with a relatively small effect. The other HD QTL on the long (bottom) arm of chromosome 3 had a small effect, explaining 5.5% of the variation in HD, and was detected only in IM analysis. Lin et al. (1998) also reported a QTL for HD in a similar location with small effect.

Relationship between QTLs Affecting Sheath Blight Resistance, Plant Height, and Heading Date

Simple correlation analysis revealed that PH and HD were significantly ($p < 0.01$) associated with SB resistance, with $r = -0.636$ and -0.216, respectively. Similar correlations have previously been reported by Li et al. (1995b). However, unlike other reports, PH and HD were not correlated with each other in this population ($r = 0.08$). A multiple regression model using PH and HD indicated that together these agronomic traits explain approximately 43% ($p < 0.0001$) of the observed phenotypic variation in SB resistance. The regression model also indicated that PH and HD individually explained 41 and 5% ($p < 0.0001$) of the variation in SB resistance, respectively. Thus, increased PH resulted in a dramatic reduction in disease severity, while delayed HD did not affect SB resistance nearly as much. Li et al. (1995b) also observed similar combined contributions of PH and HD to SB resistance, but in that study most of the variance for SB resistance was explained by HD (42%) rather than PH (4.7%). Both parents used in that study were semidwarf, resulting in a smaller effect of PH on SB resistance.

The relationship of PH and HD QTLs with SB resistance QTLs is evident from likelihood maps of LOD scores for these traits (Fig. 3). The SB resistance QTL on chromosome 1 was colocated with the QTL for PH, whereas the individual QTL LOD peaks for SB resistance and HD on chromosome 3 were separated by 6.6 to 15.3 cM (Table 2). In all cases, resistance alleles at these QTLs were associated with alleles increasing PH or delaying HD. Our results confirm previously reported associations of PH (Marchetti, 1983; Li et al., 1995a,b; Pinson et al., 2005) and HD (Marchetti and McClung, 1994; Li et al., 1995a,b; Pinson et al., 2005) with SB resistance and are contrary to the findings of Zou et al. (2000) showing no linkage of SB resistance QTL with those for PH or HD.

The association between increased PH and decreased SB incidence is not surprising, as SB disease spreads from the water line up the leaf sheath and toward the panicle, and our rating was based on the percentage of vegetative height displaying SB disease lesions. A comparatively smaller portion of tall plants would therefore be infected if the disease spreads at the same rate, irrespective of PH. Furthermore, the shortened internodes of semidwarf plants may create a more tightly closed canopy and a microclimate that is more conducive for disease development in comparison to tall plants.

In the present study, the SB resistance QTL on chromosome 3 mapped near a QTL associated with delayed HD. However, since the peak locations of these QTLs were separated, this implies that there are separate linked genes

Table 2. Positions of QTLs for sheath blight (SB) resistance, plant height and heading date determined by interval mapping (IM) and composite interval mapping (CIM).

Trait	Chr	IM					CIM				
		Position	Peak marker	LOD†	PVE‡	Add§	Position	Peak marker	LOD	PVE	Add
		cM					cM				
SB score	1	162.7	RM1339	25.2	36.4	1.44	162.7	RM1339	30.6	35.0	1.40
	2	132.4	RM3685	3.6	5.8	–0.57	133.0	RM3685	5.5	5.8	–0.58
	3	15.5	RM3117	3.9	6.4	0.61	6.6	RM7072	5.3	2.4	0.37
	9	114.2	RM3823	3.5	7.0	0.66	113.5	RM3823	5.2	7.0	0.62
Plant height	1	162.7	RM1339	52.4	77.0	–13.8	162.7	RM1339	55.1	88.9	–16.33
	9	–					34.1	RM7364	4.3	9.8	–1.24
Heading date	3	0.2	RM3894	21.3	29.7	–2.47	0.0	RM3894	24.2	28.7	–2.41
	3	143.7	RM2334	3.4	5.5	0.81	–				

†Logarithm of odds ratio.

‡Percentage of phenotypic variation explained by QTL.

§Additive effect of substituting a 'Rosemont' allele for a 'Pecos' allele in the QTL region. Note that increased SB score represents increased susceptibility in the 0 to 9 SB rating system used.

controlling these traits at this location. This finding also suggests that the correlation between SB resistance and HD may be partially due to linkage, rather than to the direct interrelation of these traits in this population. A larger progeny population size or higher density map could validate these findings. Still, these results corroborate the previous findings of Li et al. (1995a,b) that indicated the separation of LOD peaks for SB resistance and HD QTLs in a similar region on chromosome 3. Furthermore, proteomic evidence indicates that candidate disease resistance genes, such as glyceradehyde 3-phosphate dehydrogenase and chitinase, are found in this SB resistance QTL region (Lee et al., 2006).

The SB resistance QTLs on chromosomes 2 and 9 appear to be significantly independent of PH and HD. This is in partial agreement with previous studies (Li et al., 1995b; Pinson et al., 2005) that indicated a PH QTL in the same region as an SB resistance QTL on chromosome 2. The identification of these SB resistance QTLs independent of increased PH and delayed HD makes them more useful to breeders, since these traits are often undesirable in commercial cultivars.

It is worthwhile to indicate the likely sources and prevalence of SB resistance alleles we identified in evaluating the effort needed for introgressing these regions into new rice varieties, particularly for alleles in regions other than the semidwarf locus on chromosome 1. Using the data of Garris et al. (2005) and Lu et al. (2005), analyses of SSR marker diversity among rice germplasm in the SB resistance QTL regions on chromosomes 2, 3, and 9 were made and summarized (Table 3). The SSR allele haplotype around the SB resistance QTL on chromosome 2 in Rosemont is moderately common in temperate and tropical *japonica* accessions. This haplotype is especially prevalent among U.S. varieties from Texas, but is rare among *indica* accessions. The SSR haplotype at the SB resistance QTL on chromosome 3 from

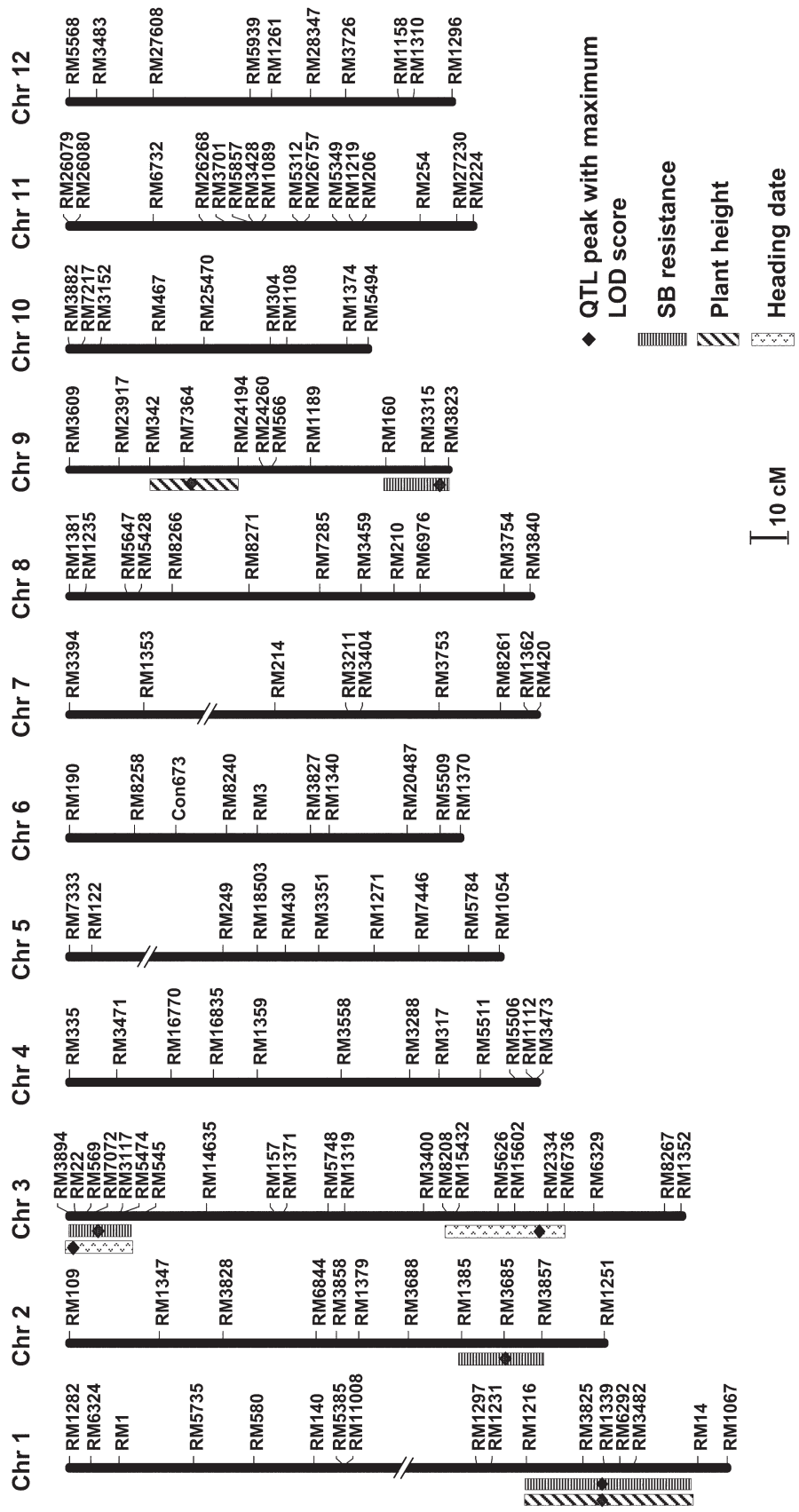

Figure 3. 'Rosemont' × 'Pecos' linkage map depicting locations of QTLs for sheath blight (SB) resistance, plant height, and heading date.

Table 3. Marker alleles for sheath blight (SB) resistance QTL regions in parents used in this study and international germplasm (data from Garris et al., 2005; Lu et al., 2005).

	Chromosome 2			Chromosome 3			Chromosome 9		
	RM106	RM221	RM250	RM22	RM231	RM489	RM1189	RM215	RM205
Position, cM†	101.5	114.0	140.7	6.0	11.5	20.3	62.0	83.0	93.5
	nt‡								
Cultivar alleles									
Rosemont	287	184	173	191	191	269	190	155	155
Pecos	293	184	177	193	191	271	180	149	157§
Tainan Iku 487	293	184	165	185	191	271	180	149	157§
Prevalent alleles¶									
Indica	287,293	191	153	193	185	235	174	147,149	123,155
Japonica									
Temperate	293	184	177	185	191	271	180	149,155	155
Tropical	287,293	184	171,173,177	185,191,193	181,183,191	269	176,180,190	151,155	123,155,161

†IRMI 2003 map position, according to Gramene database (www.gramene.org).

‡nt, nucleotide length of SSR marker allele.

§Rare allele for RM205, present in less than 2% of germplasm tested.

¶Alleles called prevalent when present in more than 20% of germplasm tested.

Pecos frequently appears in temperate *japonica* worldwide germplasm, including most U.S. medium-grain varieties from California. This Pecos haplotype is infrequent in tropical *japonica* germplasm and is rare in *indica* germplasm. The SSR allele haplotype from Pecos at the SB resistance QTL on chromosome 9 is rare in U.S. rice germplasm and represents a novel introgressed region from its parental line Tainan Iku 487, a temperate *japonica* "ponlai" type rice from Taiwan (PRC). It can be noted that the only other germplasm accession seen to carry both of the Pecos/Tainan Iku 487 SSR haplotypes on chromosomes 3 and 9 is Nortai (data not shown), which is also derived from Tainan Iku 487 and is a notable (though relatively underutilized) SB-resistant tropical *japonica* U.S. cultivar (Johnston et al., 1973).

The MIM using WinQTL Cartographer 2.5 indicated that there was no evidence for the presence of epistasis between the QTLs identified in the present study. Such lack of epistasis between QTLs has also been observed in previous studies (Stuber et al., 1987; Paterson et al., 1991; Li et al., 1995a).

CONCLUSIONS

Our identification of four QTLs for SB resistance within tropical *japonica* germplasm is a valuable finding for rice geneticists, breeders, and pathologists. In spite of the difficulties associated with quantitative traits, these SB resistance QTLs were stable across replications and years, signifying their reliability. Although most of the QTLs reported in our study explained less than 10% of phenotypic variation, they were consistent across years, in contrast with the findings of Zou et al. (2000) that found a lack of robust QTLs for SB resistance across years. The most significant of the four QTLs for SB resistance was colocated with a QTL associated with PH at the *sd-1* locus on chromosome 1.

The present study further supports the previous finding that agronomic traits, particularly PH (Marchetti, 1983; Li et al., 1995a,b; Pinson et al., 2005), have a major impact on SB resistance in rice. Identification of SB resistance QTLs are more likely to be used in breeding programs when they are independent of mechanisms of disease escape, like delayed heading and increased PH, which are considered undesirable agronomic traits. The SB resistance QTLs on chromosome 2 and 9 appear to be independent of such associations, explaining 5.8 and 7.0% of phenotypic variation for SB resistance, respectively. Additionally, although the SB resistance QTL on chromosome 3 arising from Pecos is found near an HD QTL, the genes controlling HD and SB resistance appear to map at separate loci, so the linkage between these traits appears capable of being broken. Marker-assisted introgression of the relatively uncommon SB resistance alleles from Pecos on chromosomes 3 and 9 into any of the predominantly susceptible U.S. cultivars would be expected to improve their SB resistance. Combining traditional breeding approaches with marker-assisted selection will likely facilitate development of rice cultivars having improved SB resistance.

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